three, for the same reason, as is indeed stated in the text. Even these three decades are composed of two different experiments (radar data sensitive to rainfall and satellite pictures of clouds), covering each about two orders of magnitude, with some overlap. The other two examples mentioned by Mandelbrot are temporal self-affine trails. As stated in (*2*), such trails fall outside the domain of our discussion, because the time axis can be extended at will. Moreover, the eight cases in (*1*) and (*2*) with a scaling range extending beyond two decades are dominated by spatial self-affine fractals, such as sections of rough surfaces and fronts (*8*). This further lowers the average number of decades in isotropic self-similar fractals. As in temporal self-affine trails, an experiment leading to spatial self-affinity can in principle start with as long a front as desirable and is thus not limited in scaling range.

In conclusion, it appears that the limited-range empirical fractals (*9*) are the dominant justification for "the fractal geometry of nature." Rather than sweeping them under the carpet as "bad data," their limited range should be carefully studied and understood. An intriguing and fundamental question that remains open is, Why are these limited-range fractals so common?

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9. The usefulness and authenticity of the limited range empirical fractals, which is a central theme in (*2*), is not addressed by Mandelbrot. Thus, there is no cause for alarm that "Avnir is withdrawing his earlier claims." On the contrary, not only does Avnir stand behind the usefulness of the many limited-range fractals he, P. Pfeifer, and their many coworkers have found over the years, but he continues to detect them and reported recently on a method of controlling the effective small-angle x-ray scattering surface fractality of modified silicas [C. Rottman, G. S. Grader, Y. De Hazan, D. Avnir, *Langmuir* **12**, 5505 (1996)].

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Muon Collider

Alexander Hellemans (News, 9 Jan., p. 169) conveys the physics of muon colliders to an admirable extent, and I agree with much of what is said in his article. I am an advocate of working on muon collider research and development (R&D), and I am even a subspokesman for the collaboration, for which Robert Palmer of Brookhaven National Laboratory is the spokesman. However, because of the context of certain quotes, the article conveys an inaccurate impression of some of my views.

Although I am working hard to make it a reality, I would not say, for example, "We can build a Higgs factory." My view is that the option is very attractive, but must receive strong funding support from the U.S. Department of Energy and strong R&D commitment from the community if we are to know that such a machine is a viable option at

the time that we must make a choice among the various alternatives.

Palmer, I, and the rest of the Muon Collider Collaboration, which now consists of 100 working physicists, agree in thinking that R&D money should be spent on muon colliders; in particular, both the production of muons and the cooling of the muon beam need experimental demonstration. This is the only way to find out if this exciting possibility is "real" or not. Still to be seen is whether the funding agencies agree with us.

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Insulin/IGF Signaling and HNF-3/*forkhead* Proteins

A report by Kui Lin *et al.* (14 Nov., p. 1319) and a paper by S. Ogg *et al.* (*1*) demonstrate that *daf*-16, a member of the HNF-3/*forkhead* protein family, contributes to the longevity of *Caenorhabditis elegans* when the activity of *daf*-2, a homolog of the insulin and IGF-I receptor, is compromised. These interesting studies provide strong genetic evidence that signaling by the insulin/IGF-I receptor and HNF-3/*forkhead* transcription factor homologs may exert opposing biological effects.

Both papers note that HNF-3 proteins have been found to interact with cis-acting DNA sequences that mediate inhibitory effects of insulin on the expression of multiple genes in the liver, including phosphoenolpyruvate carboxykinase (PEPCK) and insulin-like growth factor binding protein-1 (IGFBP-1), yet neither cites the first papers regarding this potentially seminal observation (*2*, *3*). In those studies, we reported that HNF-3 proteins bind to highly related insulin response sequences in both the IGFBP- I and PEPCK promoters. We also found that HNF-3 binding at this site may enhance the ability of glucocorticoids to stimulate IGFBP-1 promoter activity and demonstrated that overexpression of HNF-3β stimulates IGFBP-1 promoter activity in NIH 3T3 cells in a sequence-dependent fashion (*3*). To our knowledge, this report remains the only published data directly demonstrating that HNF-3 proteins and insulin can exert antagonistic effects on gene expression through identical or overlapping cis-acting DNA sequences.

In their report, Lin *et al.* suggest that signaling from insulin and/or IGF-I receptors may directly disrupt effects of HNF-3/*forkhead* proteins on gene expression, perhaps by reducing the binding of HNF-3/*forkhead* proteins to their target sequence. Although this may be the case, subsequent studies in this and other laboratories indicate that HNF-3 binding does not correlate with the ability of insulin to suppress either IGFBP- I or PEPCK promoter activity, but relates more directly to the ability of glucocorticoids to transactivate both of these promoters (*4*). Also, we are not aware of any data indicating that insulin disrupts the binding of HNF-3 proteins to insulin response sequences in either the IGFBP-1 or PEPCK gene. Indeed, both HNF-3 proteins and insulin have been found to contribute positively to hepatic expression of IGF-I (*5*).

In view of these findings, it may well be that the functional antagonism between the effects of the insulin/IGF-1 receptor signaling system and members of the HNF-3/*forkhead* family on longevity in *C. elegans* may reflect molecular mechanisms that are neither simple nor direct.

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